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Inhibition of tumor cell adhesion to lymph nodes by laminin-related peptide and neuraminidase.

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BACKGROUND. Adhesion to lymph nodes, rather than growth stimulation, accounted for preferential colonization of lymph nodes by a metastatic B16 melanoma. We investigated these adhesive interactions. **METHODS.** Four classes of molecules were tested for inhibition of melanoma adhesion to cryostat sections of lymph node. **RESULTS.** Calcium chelators ethylenediaminetetraacetic acid and ethyleneglycol-bis-(beta-aminoethylether)-N,N,N',N'-tetra acetic acid completely inhibited adhesion (50% adhesion, half-maximal inhibition, at 1 to 3 mmol/L). Cytochalasin B, which impairs contractile microfilaments, inhibited adhesion (60% adhesion at .001 mmol/L, 28% at .01 mmol/L). Colchicine, which disaggregates microtubules, had a similar effect (20% at .01 mmol/L, lowest dose tested). Trypsin slightly increased adhesion (125% adhesion at 10 micrograms/ml). Neuraminidase, which removed sialic acid residues, inhibited it (50% adhesion at 5 micrograms/ml). Gly-arg-gly-asp-ser, a peptide with a cell binding sequence of fibronectin, did not consistently inhibit adhesion (69% adhesion at 0.1 mg/ml, 83% adhesion at 1 mg/ml) or substantially differ from gly-arg-gly-glu-ser-pro (59% adhesion at 0.1 mg/ml, 90% adhesion at 1 mg/ml). In contrast, a peptide with a cell binding region of laminin (tyr-ile-gly-ser-arg) inhibited adhesion (50% adhesion at .05 mg/ml). **CONCLUSIONS.** Tumor cell-lymph node adhesion is a calcium-dependent process, requiring a functional cytoskeleton, that is mediated by both sialic acid moieties and trypsin-resistant, laminin-related, adhesion molecules.

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